The major plant pathogens occurring in native ecosystems of south-western Australia

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Abstract

Objective assessment of the relative importance of pathogens on conservation and production values in native plant communities of south-western Australia is impeded by the lack of systematic disease surveys. The occurrence of diseases and pathogens on Western Australian native plants was compiled from published information, other reports and personal databases. Pathogens were databased according to name, host name and family, disease group and Botanical Province, giving a total of 936 entries that did not include reports of pathogens on hosts in nurseries. Ninety-one per cent of the pathogen reports were from the South-West Botanical Province and 2% from each of the Eremaean and Northern Botanical Provinces. Bacterial diseases, galls, downy and black mildews, ergot and leaf moulds were infrequently reported on native plants. Pathogens were infrequently reported on species within the families: Aizoaceae, Amaranthaceae, Amaryllidaceae, Annonaceae, Anthericaceae, Apocynaceae, Arecaceae, Asphodelaceae, Cupressaceae, Cyperaceae, Dennstaedtiaceae, Geraniaceae, Juncaceae, Lamiaceae, Linaceae, Loganiaceae, Olacaceae, Onagraceae, Phormiaceae, Pittosporaceae, Podocarpaceae, Polygonaceae, Portulacaceae, Rubiaceae, Solanaceae, Stylidiaceae, Tremandraceae, Verbenaceae and Zamiaceae. Pythiacious root rots, rusts, Armillaria root rots, stem cankers, and leaf spots and blights were frequently reported on native plants. Families most affected by disease were: Proteaceae, Myrtaceae, Mimosaceae, Papilionaceae, Haemodoraceae, Goodeniaceae, Epacridaceae, Poaceae and Chenopodiaceae. Families mostly affected by rusts were least affected by the root rots, stem cankers and leaf spots and blights. The biology, distribution and disease expression of Phytophthora cinnamomi, rust fungi, Armillaria luteobubalina and Cryptodiaporthe canker of Proteaceae in native plant communities are described. Conservation of plant taxa requires a much better inventory, than is available at present, of the incidence and status of the various plant pathogens that occur in native communities of southwestern Australia. Prediction of the likely long-term effects of pathogens on native plant communities requires a much better understanding of their life cycles and biology in the south-western Australian environment.

Introduction

The lack of systematic disease surveys in native plant communities of south-western Australia impedes objective assessment of the relative importance of the impacts of pathogens on conservation and production values (Shearer & Hill 1989; Shearer 1992a). There have been no coordinated regional surveys of disease occurrence in native communities of south-western Australia, similar to the regular assessment of disease and pest conditions in Canadian forests (Forestry Canada 1993). This is somewhat surprising considering the exceptional species richness and high degree of endemicity of the flora of south-western Australia. At least 7000 species of described native vascular plants occur within the state (Green 1985), of which over 3000 are endemic to the area (Keighery 1992). Knowledge of the diseases of native plant taxa is important for maintenance of long-term conservation and production values, especially in the case of rare and endangered taxa.

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Current knowledge of the occurrence, biology and impact of pathogens in Western Australia has mainly accumulated from research initiated in response to potential disease threats to wood production, such as early research on wood rots and jarrah dieback (Shearer 1992b), or from opportunistic individual curiosity and observation. Since the mid 1980's, there has been a growing appreciation of the threat of disease to conservation values (Shearer 1992b).

This paper assesses the relative occurrence of pathogens on native plant species from published information and personal databases and then describes the biology, distribution and disease expression of the major pathogens affecting Western Australian native plant communities.

Diseases and pathogens of Western Australian native plants

Occurrence of diseases and pathogens on Western Australia native plants was compiled from published information and reports (Brandis *et al.* 1984; Brittain 1989; Hill 1990; Shearer 1992a; Shivas 1989; Wills 1993) and from survey and isolation databases of my own and those of S Bellgard, F Bunny and the Plant Health Service, Department Conservation and Land Management. Pathogens were databased according to name, host name and family, disease group (e.g. Pythiacious root rots, Armillaria root rots, rust, mildew, etc) and Botanical Province. Nomenclature of plant taxa follows that of Green (1985). The database consisted of a total of 936 entries, and did not include reports of pathogens on hosts in nurseries. Because of the lack of comprehensive surveys, the current information is incomplete and the database represents an indication of the occurrence of a pathogen on a particular host species rather than the intrinsic frequency of its occurrence. The number of occurrences within families can give some measure of the pathogens relative importance. The analysis will, however, favour pathogens and plant taxa most studied. The analysis will also favour pathogens with wide host range, and disfavour those with high impact but narrow host range.

Ninety-one per cent of the pathogen reports were from the South-West Botanical Province and 2% from each of the Eremaean and Northern Botanical Provinces. This partly reflects the greater concentration of research activity that has occurred in the South-West Botanical Province.

Disease groups occurring in three or more families and families within which there was three or more occurrences of

a pathogen are shown in Table 1. Bacterial diseases, galls, downy and black mildews, ergot, and leaf moulds were infrequently reported on native plants. Pathogens were infrequently reported on species within the families: Aizoaceae, Amaryllidaceae, Annonaceae, Amaranthaceae, Anthericaceae, Apocynaceae, Arecaceae, Asphodelaceae, Cupressaceae, Cyperaceae, Dennstaedtiaceae, Geraniaceae, Juncaceae, Lamiaceae, Linaceae, Loganiaceae, Olacaceae, Onagraceae, Phormiaceae, Pittosporaceae, Podocarpaceae, Polygonaceae, Portulacaceae, Rubiaceae, Solanaceae, Stylidiaceae, Tremandraceae, Verbenaceae and Zamiaceae. This list of families with infrequent disease does not simply reflect families with few species, as only 25% of the families have 5 or less species and just over a third have more than 40 species (Green 1985). The list may represent families that are relatively disease free, but plant taxa may also be included because of limited investigation of disease occurrence.

Pythiacious root rots, rusts, Armillaria root rots, stem cankers, and leaf spots and blights were frequently reported on native plants (Table 1). Families most affected by disease were: Proteaceae, Myrtaceae, Mimosaceae, Papilionaceae, Haemodoraceae, Goodeniaceae, Epacridaceae, Poaceae and

Table 1

Frequency of occurrence of pathogens within disease groups and families for which there was three or more records; less frequently occurring groups and families not included are listed in the text. Totals are for all entries in the database and do not necessarily match the row or column totals.

C		-			Disease	group					Total for all
Family	Pythiacious root rots	Rusts	Armillaria root rot	Stem cankers	Leaf spots and blights	Smuts	Wood rots	Powdery mildews	Crown rot	White rust	disease groups
Proteaceae	136	3	39	53	27						258
Myrtaceae	55		25	33	17		22	7			161
Mimosaceae	5	73	13		7						98
Papilionaceae	29	7	18		6						61
Haemodoraceae	5	27	1		6				3		42
Goodeniaceae	2	37	1							1	41
Epacridaceae	24		14		1						39
Poaceae		14			1	17					35
Chenopodiaceae		13		4	5						22
Dilleniaceae	12		4		1						17
Asteraceae		11	1					2			15
Rutaceae	7	2	2								11
Colchicaceae		10									10
Orchidaceae		9						1			10
Xanthorrhoeaceae	6		2		1						9
Casuarinaceae	4		3				1				8
Iridaceae	6		1		1						8
Rhamnaceae	1	3	2								6
Apiaceae	1	2	1		1						5
Dasypogonaceae	4		1								5
Euphorbiaceae	2		3								5
Restionaceae			2			3					5
Santalaceae			2		3						5
Sterculiaceae	3	1	1								5
Thymelaeaceae	2		3								5
Ranunculaceae		2	1					1			4
Myoporaceae		1	1								3
Zygophyllaceae		3									3
Total for all families	310	232	147	91	79	25	23	12	4	3	936

Chenopodiaceae. Frequency of occurrence of Pythiacious root rots, *Armillaria* root rots, stem cankers and leaf spots and blights were similar between families, although there was a more frequent occurrence of *Armillaria* root rot in the Mimosaceae than for the other disease groups. In contrast, frequency of occurrence of rusts between families was the inverse of that for the previously mentioned disease groups. Families mostly affected by rusts were least affected by the root rots, stem cankers and leaf spots and blights (Table 1). This is especially the case with the Colchicaceae and Orchidaceae which seem to be little affected by diseases other than rust, although this may also be due to limited research on the diseases of these plant taxa.

Within the most frequently occurring Pythiacious root rots (Table 1), *Phytophthora cinnamomi* Rands accounted for 54% of reports, *P. megasperma* Drechsler for 21% and *P. citricola* Sawada for 13%. Within the rusts, 53% were *Puccinia* sp. and 32% *Uromycladium tepperianum* (Sacc) McAlpine gall rust of *Acacia* species. Only one confirmed *Armillaria* species, *A. luteobubalina* Watling & Kile, is known to cause Armillaria root rot in Western Australia (Kile *et al.* 1983). Of the 17 stem canker pathogens recorded, *Botryosphaeria* sp. was the most frequent (45% of reports) followed by *Zythiostroma* sp. (14%) and *Cryptodiaporthe* sp. (11%). Of the 46 leaf spot and blight pathogens recorded, three species were the most frequently recorded, with each only 6% of reports.

Biology of major pathogens

Knowledge of the life cycle and biology of *P. cinnamomi* in native communities of south-western Australia is derived mainly from research conducted in the northern *Eucalyptus marginata* Donn ex Smith forest (Dell & Malajczuk 1989; Shearer & Tippett 1989), but relatively little specific information is known of the biology of *P. cinnamomi* in non-forest communities. In addition, little specific information is known of the factors affecting spore production, infection and host susceptibility to infection for *Phytophthora* species other than *P. cinnamomi*, rusts, *A. luteobubahina* and stem cankers in the south-western Australian environment.

Phytophthora root rots

Phytoplithora spp. are introduced soil-borne pathogens belonging to the class Oomycota, a relative primitive group of fungi having a number of morphological, physiological and biochemical characteristics found in certain protozoa and bacteria and an ancestral affiliation with heterokont algae (Barr 1983). In evolutionary development, *Phytophthora* belongs to a transitional group between entirely aquatic and completely terrestrial fungi. This is reflected in their complex life cycles dependent on moist conditions for survival, sporulation, dispersal and infection, and in the initiation of various adaptation strategies to cope with the fluctuating soil environment.

Phytophthora cinnamoni is a major pathogen in the alternating temperature and moisture mediterranean climate of south-western Australia, despite the fact it is an introduced, moisture-dependent microorganism. This has occurred because movement of infected soil by human activity has spread the pathogen throughout the region (see below). In addition, the soils and topography in conjunction with the hydrological cycle and susceptible plant communities have provided niches within the soil profile whereby *P. cinnamonii* can survive adverse conditions, and be spread in water or by root-to-root contact to infect the roots of a wide range of hosts. The interactions that have created the diversity of microenvironments and conditions favourable for sporulation, survival, dispersal and infection are detailed in Shearer & Tippett (1989) and can only briefly be described here.

Phytophthora cinnamomi takes advantage of favourable warm and moist soil conditions in autumn and spring, and presence of susceptible tissue, by rapidly producing various spore types in an expanding phase of population growth. During unfavourable conditions of low soil moisture, absence of susceptible tissue, and high microbial activity, the fungal hyphae are lysed and disintegrate, releasing resistant spores specialised for survival. Vegetative reproduction is by sporangia that release infectious motile zoospores in water. This is the main way the Phytophthora species reproduce and infect plants. Spherical, sedentary chlamydospores may also be vegetatively produced, but their role in infection and survival in south-western Australia is poorly understood. Under certain conditions, sexual reproduction by thick-walled oospores occurs. Oospore production by P. cinnamomi is probably infrequent in south-western Australia as two mating types are required for spore induction but only one mating type predominates in the region. In comparison, P. citricola and P. megasperma readily produce oospores from the one mating type. Reproduction by oospores is probably an important survival mechanism for P. citricola and P. megasperina as the thick walled spores are more resistant to drying than are zoospores.

Once *Phytophthora* species have entered the roots of susceptible hosts, primary symptoms of infection are evident as advancing fronts of necrosis (lesions) in the inner bark of roots and stems. Lesions are most evident in fleshy primary roots as a root rot. The fungi kill their hosts by destroying the roots and girdling the base of the stem, depriving the plant of access to nutrients and water. Host plant species occur mainly in the Proteaceae, Myrtaceae, Papilionaceae, Epacridaceae and Dilleniaceae (Table 1).

Rusts

Although rusts are the second most frequent pathogens on native plant taxa in south-western Australia (Table 1), research on their biology in the region is limited to only three studies (Goodwin 1963; Verhoogt & Sivasithamparam 1985; Nichol 1986). Rust fungi are of the order Uredinales of the class Basidiomycota and are destructive pathogens to many agriculture and forest crops. In contrast to *Phytophthora*, rust pathogens on native plants are probably endemic, they complete their life cycles on the above ground plant parts and they are mainly dispersed as air borne spores. Also, unlike root rots and stem cankers which can live and reproduce on dead tissue, the rust fungi are obligatory parasitic, requiring living hosts for normal development.

The life cycles of rusts are more complex than those found in any other group of fungi, and typically consist of four or five reproductive stages in a regular sequence. Details of the stages can be found elsewhere (Agrios 1978) and are briefly described as follows. Pycniospores and receptive hyphae are

produced in pycnia. Pycniospores serve as spermatia and are transferred to other pycnia by insects and fuse to form binucleate hyphae. Aeciospores formed from the binucleate hyphae are wind-dispersed to infect hosts other than the one on which they are produced. Uredospores are produced from binucleate mycelium from a germinating aeciospore or a uredospore. Uredospores are generally the main repeating stage of rusts and can withstand adverse conditions of longrange dispersal from plant to plant by wind. Sexual reproduction is by teleutospores, which are not dispersed but germinate to produce basidiospores. The basidiospores are temperature and moisture sensitive, and dispersed by wind over short distances. Within this life cycle pattern, longcycled rusts produce at least one type of binucleate spore in addition to the teleutospore, while for short-cycled rusts the teleutospore is the only binucleate spore produced. The life cycle may be completed on the one host (autoecious) or on two distinct hosts (heteroecious).

In Western Australia, gall rust (*Uromycladium tepperiauum*) is a short-cycled autoecious rust producing pycniospores, teleutospores and basidiospores mainly on Acacia species (Goodwin 1963). Some rust taxa on orchids are long-cycled as they produce aeciospores, uredospores and teleutospores (Nichol *et al.* 1988). It is not known whether the life cycle of rusts on plant taxa other than the Mimosaceae (Table 1) are autoecious or heteroecious.

Host plant species occur mainly in the Mimosaceae, Goodeniaceae, Haemodoraceae, Poaceae, Chenopodiaceae, Asteraceae, Orchidaceae and Colchicaceae (Table 1). Uromycladium tepperianum infection stimulates the Acacia host to form galls and / or 'witches brooms' (Goodwin 1963). Infection of the growing point results in a witches broom caused by reduction of the growing axis and a proliferation of lateral buds. Galls may be globose or elongated and can form on different plant organs, although formation on a particular plant part is consistent within an Acacia sp. (Goodwin 1963). Gall formation on inflorescences reduces fertilisation and fruit development. Uredospore production by leaf rusts rupture the leaf epidermis, reducing photosynthetic and transpiration processes. In Orchidaceae, leaves infected with rust senesce earlier than healthy leaves and rusted plants produce fewer flowers than healthy plants (Nichol 1986).

Armillaria root rot

Research on *A. luteobubalina* in south-western Australia has mainly concentrated on the impact of the pathogen in forested areas (Pearce *et al.* 1986; Shearer & Tippett 1988; Pearce & Malajczuk 1990a), and the potential for biocontrol with wood decay fungi (Pearce & Malajczuk 1990b). The impact of the pathogen on shrubland and heathland communities has only recently been recognised (Shearer *et al.* 1994). Specific details are lacking on the mechanisms of infection and host colonisation of *A. luteobubalina* in Western Australia and factors affecting host susceptibility to infection (Shearer 1992a). Details of the life cycle of *Armillaria* species are reviewed in Shaw & Kile (1991).

Armillaria luteobubalina is an indigenous species of mushroom-producing primary pathogen of the order Agaricales, class Basidiomycota. Infection from *A. luteobubalina* occurs from aerial dispersed basidiospores or through mycelial transfer at root contacts. Growth through the soil by rhizomorphs is not an important mechanism of spread in south-western Australia (Pearce et al. 1986; Shearer & Tippett 1988) as the seasonal pattern of temperature and moisture associated with the mediterranean climate of the region, is not conducive for rhizomorph growth (Pearce & Malaiczuk 1990c). Basidiospores, formed by sexual recombination of gametes, are shed in autumn-winter from annual fruiting bodies that develop on decayed roots and stems of dead and living trees. Fruiting bodies of A. luteobubalina are mainly produced in June and July (Pearce et al. 1986; Shearer & Tippett 1988). How basidiospores infect woody tissue is poorly understood and is probably an infrequent event (Kile 1983). The distribution of infection points and aerial dispersed sexually produced basidiospores results in a discontinuous, discrete distribution of infections of different genotypes. The number and distribution of different genotypes can provide an estimate of the frequency of infection from basidiospores (Kile 1983), but no analysis of this type has been done for south-western Australia. The pathogen spreads within disease centres by mycelial growth through roots. In susceptible E. wandoo Blakely, the mean rate of disease extension over a 8 year period was 2.04 ± 1.05 m yr⁻¹ (Shearer unpub. obs.). This is comparable to mean maximum rates of 0.7-1.6 m yr⁻¹ found by Kile (1983) for Victorian forest. New infections are established by contact between roots and stems, and dead roots and stumps increase the inoculum level. In mixed eucalypt forests in the highlands of westcentral Victoria, the pathogen can survive in stumps for up to 30 years (Kile 1981).

Armillaria luteobubalina establishes in the bark and causes columns of decay within roots and stems of host species. The pathogen spreads tangentially in the inner bark of susceptible hosts, often resulting in girdling of the stem collar and host death (Pearce *et al.* 1986; Shearer & Tippett 1988). Host plant species occur mainly in the Proteaceae, Myrtaceae, Papilionaceae, Epacridaceae and Mimosaceae (Table 1).

Stem cankers

The contribution of canker fungi to stem and branch death in south-western Australia has largely been ignored (Davison & Tay 1983; Shearer 1992a). Mortality and decline of marri and red flowering gum were associated with stem cankers in the mid 1930's (Smith 1970). Davison & Tay (1983) identified a number of pathogenic fungi associated with stem and branch cankers of forest trees in south-western Australia. In 1989, a species of Diplodina (sexual stage Cryptodiaporthe) was found killing Banksia coccinea R. Brown on the south coast of the state (Shearer & Fairman 1991). Interpretation of the cause of stem cankering can be complicated as some fungi are frequently isolated from cankers but they are secondary invaders of the diseased tissue. Cytospora eucalypticola van der Westhuizen is an example of a frequently isolated fungus that pathogenicity tests have shown to be a nonagressive facultative parasite (Davison & Tay 1983; Shearer et al. 1987).

The origins of stem canker fungi in south-western Australia are uncertain. *Botryospluaeria ribis* Gossenb. & Dugg. is possibly an introduced pathogen (Davison & Tay 1983) and it is widely distributed on a diverse range of hosts in the tropical and temperate regions of the world. The *Cryptodiaporthe* pathogen of *B. coccinea* is possibly endemicas it is a new species (Bathgate *et al.* 1994) and has a very limited host range within the Proteaceae (see below).

How the canker-causing fungi complete their life cycles in south-western Australia requires further research. This is complicated by uncertainties in the identity of the various spore stages of canker fungi on native plants in this state. For example, the asexual stage of *Endothia* isolated from Myrtaceae in Western Australia has been identified as *En. gyrosa* (Schw. Fr.) Fr. by isozyme analysis against voucher specimens (Davison & Coates 1991). Even though the sexual ascospore stage occurs in eastern Australia (Walker *et al.* 1985), it has yet to be recorded in Western Australia.

Canker fungi kill the aerial parts of plants. This is in comparison to disease caused by Phytophthora and Armillaria that kill plants from the roots up. Hosts affected by canker fungi occur mainly in the Proteaceae and Myrtaceae (Table 1). The fungi sporulate in dead bark and are dispersed as sexually produced ascospores in wind currents or asexually produced pycnidiospores in rain splash. The mode of entry of germinating spores is either direct or gained through lenticels or wounds from branch stubs, broken branches and insect damage. Phloem and sapwood invasion results in sunken cracked areas on the stem that may expose the xylem and exude kino. Cankers thus formed can be annual, perennial or diffuse. In annual cankers, lesion development is contained by host defense mechanisms within the first year's invasion. Botryosphaeria ribis generally forms annual cankers unless stress factors affect the host-pathogen interaction, as described in the next section. Perennial cankers denoted by concentric rings are formed when invasion by the pathogen is walled off, but the pathogen survives on dead tissue to reinvade healthy tissue in the following years. Large 'target'like cankers occur on E. calophylla Lindley and E. gouphocephala DC, but the causal pathogen has yet to be determined. Diffuse cankers occur when lesions rapidly progress along the stem, resulting in gradual decline from death of twigs and lateral branches to rapid death of leaders in a few years. Diffuse canker development by Cryptodiaporthe sp. leads to death of infected B. coccinea, and destruction of diseased stands in a few years (Shearer & Fairman 1991; see below). The effect of death of canker-infected stems and branches on leaf area and host plant functioning has not been determined.

Disease caused by canker fungi can be aggravated by transient stress factors (Schoeneweiss 1975). Trees planted outside the normal range may experience environmental stress with an associated decline in resistance to infection by canker organisms (Shearer *et al.* 1987). Stress from two days of above 40 °C and high winds in February 1991, was associated with rapid extension of *Bo. ribis* lesions in stems of *B. speciosa* R Brown near Hopetoun. The stand was severely debilitated by the infection and trees died. Twelve months later, many of the surviving *B. speciosa* trees had again contained the *Bo. ribis* lesions and formed new epicormics below the walled-off lesion margin.

Distribution

Phytophthora cinnamomi

Phytophthora cinnamomi is the most common and destructive of the *Phytophthora* species found in native communities of the south-west. It occurs in the area bounded by Eneabba north of Perth, east of Dryandra near Popanyinning, and Cape Arid east of Esperance on the south coast (Fig 1).

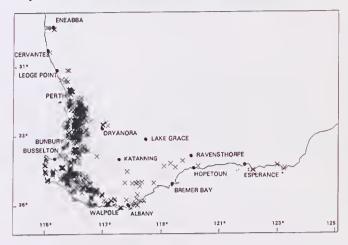


Figure 1. Distribution of *Phytophthora cinnamomi* disease centres in south-western Australia, compiled mainly from assessment plots and mapping, and supplemented by isolation records.

Greatest incidence of *P. cinnamomi* occurs in the northern and southern *E. marginata* forest (Fig 1). This is partly due to environment and partly to historical factors related to human activity (Shearer & Tippett 1989; Shearer 1992a). The pathogen frequently occurs on the acidic leached sands of the Bassendean Dune System of the Swan Coastal Plain, Gavin Sands of the Leeuwin-Naturaliste Ridge, laterite soils and winter wet flats of the d'Entrecasteaux and Walpole-Nornalup National Parks and the Keystone and Gardner geomorphic units in areas on the south coast such as West Cape Howe and Two Peoples Bay. Incidence is high in the sandy deposits of the Stirling Range National Park (the rectangle of occurrences north of Albany, Fig 1). Infections fringe the Fitzgerald River National Park east of Bremer Bay, but a 6 km long infection occurs within the park.

A transect from the coast, inland between 31.5° and 33.5° S, shows that *P. cinnamomi* disease centres are absent from coastal dunes, but increase in frequency in the Bassendean Dune System and Pinjarra Plain to the west of the Darling Scarp (Fig 2). Frequency of occurrence is greatest in the northern *E. marginata* forest on the western edge of the Darling Scarp, decreasing rapidly to the drier eastern edge of the *E. marginata* forest (Fig 2).

Rusts

Rusts are widely distributed on native plant taxa throughout the south-west (Fig 3). This is especially so for *U. tepperianum*, which occurs relatively frequently on *Acacia* spp. in coastal areas and in the eastern wheatbelt and goldfields. *Uromycladium tepperianum* is probably the most widely distributed pathogen in native communities in south-western Australia (compare Fig 3 with Figs 1 and 4-6). However, because of the limited research on rusts of native plants of Western Australia, many more surveys are needed for a more accurate picture of the distribution of rusts in native communities of the state.

Armillaria luteobubalina

Armillaria luteobubalina disease centres mainly occur in coastal dune vegetation and forested areas (Fig 4). In vegeta-

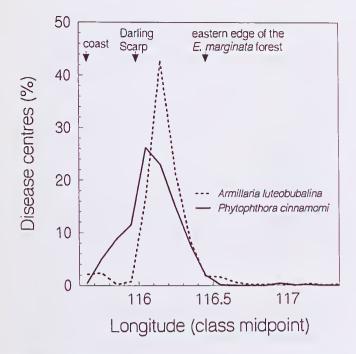


Figure 2. Occurrence of disease centres of Armillaria luteobubalina and Phytophthora cinnamomi in a transect between 31.5° and 33.5° S and from the coast (115.6° E) inland to 117.4° E. The plot is percentage of occurrence of disease centres in longitude classes of one tenth of a degree.

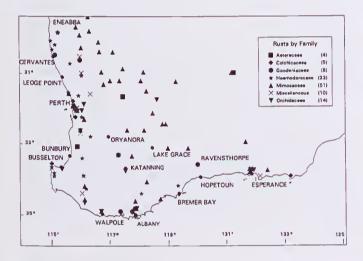


Figure 3. Distribution of rusts on native plants in south-western Australia, compiled from reports in Shivas (1989) and Nichol(1986). Rust taxa were: *Puccinia* for Haemodoraceae, *Uromycladium* for Mimosaceae, *Aecidium* and *Puccinia* for Asteraceae and Colchicaceae, and *Puccinia* and *Uromyces* for Goodeniaceae and Orchidaceae.

tion on the non-podsol sands of the coastal dunes, *A. luteobubalina* occurs as far north as Cervantes and around the coast to Cape Arid (Fig 4). The pathogen also occurs in *E. gomphocephala* forest and *Banksia* woodland of the Spearwood Dune System and equivalents, just inland from coastal dunes but rarely occurs in communities on the acid sands of the Bassendean Dune System. The pathogen frequently occurs in the northern and southern *E. marginata* forest, the *E. diversicolor* F. Muell. forest in the south, and in *E. wandoo* forest to the east.

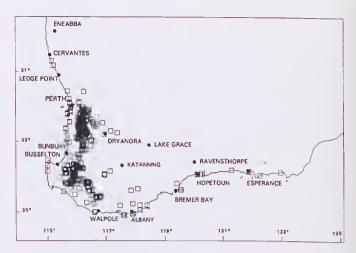


Figure 4. Distribution of *Armillaria luteobubalina* disease centres in south-western Australia, compiled from isolation records, assessment plots and mapping.

In comparison to *P. cinnamomi, A. luteobubalina* occurs on the coast and rarely occurs in the Bassendean Dune system to the west of the Darling scarp (Fig 2). Distribution within the northern forest tends to be more skewed to the east, than for *P. cinnamomi*, and there is a greater frequency of occurrence in the *E. wandoo* forest east of the *E. marginata* forest (Fig 2).

Stem canker pathogens

Various canker pathogens, mainly on Myrtaceae and Proteaceae, are widely distributed throughout the southwestern Australian region (Fig 5). The distribution map is incomplete, however, as there has been inadequate sampling in the eastern wheatbelt and goldfields.

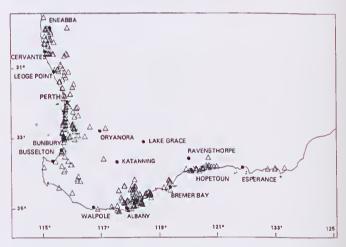


Figure 5. Distribution of stem canker fungi on Proteaceae and Myrtaceae in south-western Australia, compiled from isolation records.

The recently discovered *Cryptodiaporthe* canker of Proteaceae has an interesting discontinuous distribution (Fig 6). On the south coast, the pathogen infects *B. coccinea* throughout its geographic range (Fig 6). However for *B. grandis* Willd. and *Dryandra sessilis* (Knight) Domin, *Cryptodiaporthe* canker only occurs within a small portion of the geographic range of these two hosts. The pathogen has not been found in an area between the south coast and west coast (Fig 6), even though the area has been sampled (Fig 5). On the west coast, *Cryptodiaporthe* is an aggressive canker of *D. sessilis* north of Perth and on *B. grandis* south of Perth (Fig 6). Curiously, it has been infrequently isolated from these two hosts in other areas, even though these species occur and have been sampled throughout the south-west. Possible causes of this distribution are currently under investigation.

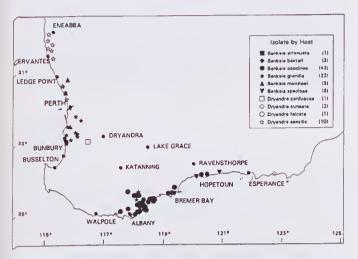


Figure 6. Distribution of *Cryptodiaporthe* stem canker of Proteaceae in south-western Australia, compiled from isolation records.

Communities affected

Phytophthora cinnamomi

Death of the susceptible understorey species of the Proteaceae, Myrtaceae, Papilionaceae and Epacridaceae (Table 1) is the first indication that *P. cinuamomi* has spread into a new area. On sites favourable to disease development, a line of dead and dying understorey marks the 'infection front' at the boundary of infested and uninfested areas (Fig 7). Disease impact is more subtle on less favourable, free draining sites, and there is often no clear demarcation between infested and uninfested areas.



Figure 7. High impact of *Phytophthora cinnamomi* in *Banksia* woodland on the Bassendean Dune System of the Swan Coastal Plain. Most of the overstorey of *Banksia attennata* R Brown, *B. ilicifolia* R Brown and *B. menziesii* R Brown has died in the infested area. Death of dominant key overstorey and understorey species result in reduction of vegetation biomass in the diseased area and the disease front being delineated by a sharp boundary of dying plants.

Shearer (1990) described the impact of *P. cinnamoni* according to a grouping of vegetation systems of Beard (1981). Impact of *P. cinnamoni* tends to be lowest in coastal communities on coastal limestone and forest communities on relatively fertile red earths associated with major valleys. Impact of *P. cinnamoni* is also low in inland woodlands and shrublands. However, low disease expression in inland areas is probably due to low rainfall unfavourable for pathogen survival and sporulation, rather than a lack of susceptible vegetation or soil profile characteristics favourable for pathogen development (Shearer 1990). This is illustrated by the recently observed infection of rare and endangered *B. cuneata* A S George, located east of Dryandra on the western edge of the wheatbelt (Fig 1).

Impact of P. cinnamonii is highest in the E. marginata forest understorey on laterites and Banksia woodlands associated with leached sands and laterites of the Northern and Southern Sandplains and the Swan Coastal Plain. Within these vulnerable communities, the impact of P. cinuanomi results more in changes in community structure and function than in total number of species. For example, infestation of Banksia woodland on the Swan Coastal Plain resulted in an average of 7 fewer species in infested than non-infested woodland (Shearer & Dillon 1994). However, the loss of these species often resulted from the almost complete death of the dominant susceptible overstorey and understorey vegetation with a substantial reduction of the vegetation biomass in the diseased area (Shearer & Dillon 1994; Fig 7). Thus in communities dominated by rare and endangered plant taxa, such as B. brownii Baxter ex R Brown in the Albany region, infestation is resulting in elimination of the threatened taxa. Keighery (1992) lists 6 species (2 Andersonia spp., B. brownii, 2 Dryandra spp. and Lambertia orbifolia C Gardner) that are currently threatened with extinction from P. cinnamomi infestation. All of these species of Proteaceae occur in the Southern Sandplains, in areas of high impact of P. cinnamouni. Within areas where P. cinnanomi has caused significant damage to susceptible communities, such as on the Swan Coastal Plain, the E. marginata forest and a number of reserves and national parks on the south coast such as Stirling Range National Park, Cape Arid National Park and Two Peoples Bay Nature Reserve, the main challenge is the development of suitable management strategies for communities irreversibly changed by impact of the pathogen.

Rusts

There is little information available on the impact of rusts in native plant communities of south-western Australia. The impact of rust on native communities of the state cannot accurately be assessed from the current information. Severe infection of *U. tepperianum* ultimately results in death of the host (Goodwin 1963) and the pathogen has been used in biological control of *Acacia saligna* (Labill)HL Wendl, a weed in South Africa (Morris 1991). In Orchidaceae, rust infection reduced the capacity of *Thelymitra crinita* Lindley to produce flowers (Nichol 1986). Thus, rust found on rare and endangered *T. macmillanii* F Muell would need to be considered in conservation plans, as seed production may be reduced by infection (Nichol 1986).

Armillaria luteobubalina

The impact of *A. luteobubalina* disease centres can be expressed as: 1) an expanding patch of dead and dying hosts;

2) dead hosts occurring frequently, but at random, in patches; 3) dead hosts occurring infrequently, but individually, or at random in patches; and 4) small patches of dead and dying hosts occurring in young stands, but the patches of mortality fail to expand as the stand ages. The first and second impact type mainly occur in coastal dune vegetation (Fig 8) and E. wandoo forest (Fig 9). The disease centres can be quite large, averaging 1.7 ± 0.2 ha (range 0.02 - 6.5 ha) for coastal dune vegetation (Shearer et al. 1994) and 1.2 ± 0.3 ha (range 0.01 - 8 ha) for E. wandoo forest (Shearer uupub. obs.). Most of the susceptible hosts are killed within the disease centres of coastal dune and E. wandoo communities, leaving open denuded areas which encourage severe wind erosion of coastal dunes (Fig 8A). In coastal dunes, geographically restricted Callitrispreisii Miq (Fig 8B) and rare and endangered B. hrownii and B. occidentalis R Brown formosa Hopper are threatened by infection. In the E. wandoo forest of the Stirling Range National Park, A. luteobubalina infestation is killing Choretrum glomeratum R Brown, the only food plant for the larvae of the rare brown azure butterfly (Wills & Kinnear 1993). The third and fourth impact type mainly occur in E. diversicolor, E. gomphocephala and E. marginata forests.

Stem canker pathogens

Stem canker pathogens are having considerable impact in communities dominated by Proteaceae and Myrtaceae in south-western Australia. *Cryplodiaporlle* stem canker is causing high mortality of *B. coccinea* (Fig 10) throughout the Banksia's geographic range on the south coast (Fig 6). In one monitored site, plant death increased from 40% to 98% in 2.7 years. The pathogen is also causing severe branch and stem cankering of *D. sessilis* north of Perth and *B. graudis*, south of Perth. On the south coast, a *Zylhiostroma* sp. causes stem cankers of *B. baxteri* and *Bo. ribis* infection has debilitated stands of *B. speciosa* in association with climatic stress.

In eucalypt forest communities, stem canker fungi are associated with crown decline, stem cankering and mortality of *E. ficifolia* F Muell, *E. calophylla* and *E. gomphocephala*. In each case, the causal pathogen has yet to be identified, although *En. gyrosa* and *Bo. ribis* have been isolated from dying *E. gomphocephala* (Shearer *unpub. obs.*). Canker fungi have been associated with the complex of factors causing crown decline in *E. waudoo* (Albone 1989). Cankers are also having an impact on myrtacious dominated communities, other than forest. For example, a *Phomopsis* sp. was isolated from dying branches of *Calothannus quadrifidus* R Brown showing severe canopy decline throughout the northern sandplain in 1993 (Shearer *unpub. obs.*).

Conclusions

Functional diversity and dynamic balance in native ecosystems result in explosive epidemics of disease being uncommon and limited in space and time (Zadoks & Schein 1979). Why then are such explosive epidemics of *Phylophthora* species, *A. luteobubaliua* and *Cryptodiaporthe* canker of Proteaceae currently occurring in plant communities of southwestern Australia? *Phytophthora* species are human introduced pathogens to native plant ecosystems of south-western Australia and their impact is related to the intensity of human activity, occurrence of sub-surface soil moisture and temperature conditions that favour survival, multiplication



Figure 8. High impact of *Armillaria luteobubalina* in coastal dune vegetation. A, Death of dune vegetation in an infested area has resulted in denuded areas subject to wind erosion in Yalgorup National Park; B, Most *Callitris preisii* have died in a disease centre on Garden Island.



Figure 9. High mortality of hosts in an Arwillaria luteobubalina disease centre in Eucalyptus wandoo forest near Kojonup.

and spread of the pathogen and large numbers of susceptible key plant taxa that have not co-evolved with the pathogens. Research has elucidated many of these interactions for *P. cinnamomi* (Shearer & Tippett 1989), but a greater understanding is required for other *Phytophthora* species such as *P. citricola* and *P. megasperma*. In contrast to *Phytophthora*, the situation for *A. luteobuhalina* and *Cryptodiaporthe* canker of Proteaceae is different, as they are probably native patho-



Figure 10. A stand of *Banksia coccinea* at Cheyne Beach, east of Albany, killed by *Cryptodiaporthe* canker. Mortality within this stand increased from 40% in 1989 to 98% in 1992.

gens and presumably have co-evolved with the existing vegetation communities. Current knowledge is inadequate to determine whether the prevailing impacts observed relate to a periodic change in disease intensity, or whether they represent more permanent long-term changes.

Conservation of plant taxa requires a much better inventory, than is available at present, of the incidence and status of the various pathogens that occur in native communities of south-western Australia. As noted in this paper, the recording of most pathogen occurrences on native plants in this state is the result of opportunistic research, and comprehensive surveys have yet to be attempted. Pathological research of native plant taxa has tended to be dominated by P. cinnamomi to the exclusion of other pathogens. Comprehensive surveys would ensure objective assessment of the importance of hitherto ignored pathogens or pathogen/ community interactions. This is illustrated by the recent recognition of the high impact of A. luteobubalina in coastal communities (Shearer et al. 1994) and Cryptodiaportlie canker of Proteaceae (Shearer & Fairman 1991; Bathgate et al. 1994). Presumably, these pathogens have been impacting on the respective communities well before their recent recognition. Biogeographical surveys of fauna and flora in communities or National Parks need to include a census of fungi occurring within the areas.

Uncertainties in the taxonomy of fungi in this state complicate inventory of the occurrence and importance of pathogens on native plants (Shearer 1992a). A number of pathogens are undescribed species. This is further complicated by the occurrence of biological species within species complexes, such as may be occurring in *P. megasperma* (Bellgard *et al.* 1994). Fungal taxonomic studies are fundamental to assessment of the relative importance of pathogens.

Prediction of the likely long-term effects of pathogens on native plant communities requires a much better understanding of their life cycles and biology in the south-western Australian environment than is available at present. By their impact, pathogens are undeniably affecting the evolution of plant communities of the state. However there is only a conceptual understanding of the selection pressures pathogens are placing on community composition and functioning, and in turn, the selection pressures environment and community composition are placing on the pathogens. Information on the biology and ecology of pathogens in native communities is needed to determine whether current impacts of endemic pathogens are short term perturbations or part of long term cycles in pathogen-community-environment interactions. Such information is also essential to the determination of the likely consequences of disease, and the application of appropriate control strategies.

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